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Anxiety Sensitivity as a Moderator of the Relation Between Trauma Exposure Frequency and Posttraumatic Stress Symptomatology

Matthew T. Feldner
University of Arkansas–Fayetteville

Sarah F. Lewis
Marshall University
Huntington, WV

Ellen W. Leen-Feldner
University of Vermont
Burlington, VT

Paula P. Schnurr
Veterans Affairs Medical Center
White River Junction, VT
Dartmouth Medical School
Hanover, NH

Michael J. Zvolensky
University of Vermont
Burlington, VT

The present study tested if the global anxiety sensitivity construct and its constituent factors (i.e., physical, mental incapacitation, and social concerns) moderate the relation between traumatic event exposure frequency and posttraumatic stress symptomatology. Participants were 61 rural young adults who reported experiencing at least 1 lifetime traumatic event. Consistent with prediction, anxiety sensitivity total and subfactor levels moderated the relation between trauma exposure frequency and posttraumatic stress symptomatology. These moderating effects were above and beyond variance accounted for by the respective anxiety sensitivity and stress main effects as well as other theoretically relevant factors (e.g., negative affectivity). Findings are discussed in relation to better understanding cognitive-based individual difference factors associated with posttraumatic stress symptomatology.

Keywords: anxiety sensitivity; trauma; posttraumatic stress disorder; moderating effects

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Anxiety sensitivity (AS), defined as the fear of anxiety and anxiety-related sensations (Reiss & McNally, 1985), is a trait-like cognitive characteristic that can predispose individuals to the development of anxiety-related problems. For example, if a person believes bodily sensations are a sign of imminent personal harm or threat, this "high AS" individual would experience escalating levels of anxiety and perhaps a panic attack when exposed to such sensations. The global AS construct encompasses fears of physical, mental incapacitation, and social experiences (Zinbarg, Barlow, & Brown, 1997), all of which can theoretically amplify preexisting anxiety (Reiss, 1991). Since the late 1980s, separate lines of research have generally supported the AS model of panic disorder vulnerability (see Taylor, 1999). Furthermore, recent studies suggest an association between AS and psychopathology beyond panic disorder, including various types of substance use disorders (Otto, Safren, & Pollack, 2004; Stewart, Samoluk, & MacDonald, 1999; Zvolensky, Schmidt, & Stewart, 2003), major depressive disorder (Otto, Pollack, Fava, Uccello, & Rosenbaum, 1995; Taylor, Koch, Woody, & McLean, 1996), chronic pain (Asmundson & Norton, 1995), hypochondriasis (Watt & Stewart, 2000), and more recently, posttraumatic stress disorder (PTSD; Taylor, 2003).

Interest in AS and PTSD has emerged, at least in part, due to the increased recognition that cognitive factors play an important role in the nature of the disorder (e.g., Ehlers & Clark, 2000). Although numerous cognitive variables have been studied in relation to the etiology and maintenance of PTSD symptoms (e.g., neuroticism; Breslau, Davis, Andreski, & Peterson, 1991; see also Schnurr & Vielhauer, 1999), AS may hold considerable explanatory promise. For example, persons high in AS who have experienced a trauma may interpret symptoms of PTSD as personally harmful (e.g., "I'm dying," "I'm going crazy"), thereby exacerbating affective symptoms (Fedoroff, Taylor, Asmundson, & Koch, 2000). As a second illustrative example, persons high in AS may be more likely to cognitively avoid trauma-related cues, thereby preventing emotional processing of the event (Lang, Kennedy, & Stein, 2002) and perhaps recovery from a traumatic event (see Brewin, Dalgleish, & Joseph, 1996). Both of these accounts are supported by previous work that has shown AS is related to catastrophic thinking (e.g., Donnell & McNally, 1990) and avoidance of emotionally salient events (e.g., Zvolensky & Forsyth, 2002) in non-PTSD relevant studies.

Although limited in overall scope, several converging pieces of empirical evidence support the postulation that AS [as measured using the Anxiety Sensitivity Index (ASI; Reiss, Peterson, Gursky, & McNally, 1986)] is related to posttraumatic stress symptomatology. First, there have been at least three studies indicating AS is elevated among persons who have experienced a trauma and developed PTSD symptoms relative to persons without such symptoms (i.e., between group tests). In the first study in this domain, Taylor, Koch, and McNally (1992) found AS was elevated among persons diagnosed with PTSD ($M_{\text{ASI total}} = 31.6$) relative to persons with no anxiety disorder ($M_{\text{ASI total}} = 17.8$). In a separate investigation, 51 survivors of a motor vehicle accident or nonsexual assault were examined. Anxiety sensitivity was significantly elevated among those with subclinical ($M_{\text{ASI total}} = 30.6$) to clinical ($M_{\text{ASI total}} = 25.2$) levels of acute stress disorder (ASD) relative to survivors without ASD ($M_{\text{ASI total}} = 12.3$; Bryant & Panasetis, 2001). Finally, Lang and colleagues (2002) recently compared women who experienced intimate partner violence (IPV) with a lifetime history of PTSD, women with a history of IPV but no PTSD, and women with no trauma history in terms of ASI total and factor scores. Total scores on the ASI were significantly higher for the trauma and PTSD group ($M_{\text{ASI total}} = 30.7$) than both of the other groups, and the no-trauma PTSD group ($M_{\text{ASI total}} = 17.0$) had higher ASI total scores than the no-trauma group ($M_{\text{ASI total}} = 7.1$). Additionally, the trauma and PTSD group endorsed significantly higher scores on the mental incapacitation, physical, and social concerns factors of the ASI than the no-trauma group. The PTSD group also endorsed higher mental incapacitation and physical concerns than the trauma-no PTSD group. The trauma-no PTSD group had higher scores only on the Physical Concerns factor than the no-trauma group.

A second line of work supporting an AS-PTSD association has evaluated the extent to which this cognitive factor relates to PTSD symptomatology (i.e., within subject tests). In an investigation of motor vehicle accident survivors, Fedoroff and colleagues (2000) found ASI total scores were significantly associated with (1) PTSD symptom severity following the accident and (2) reductions in PTSD symptoms post-PTSD treatment. Specifically, persons higher in AS reported more severe PTSD symptoms following the accident and reductions in AS were associated with reductions in PTSD symptoms following a 12-week cognitive behavioral PTSD treatment program. In a study of 121 persons reporting to a tertiary-care rehabilitation program for persons who had sustained a work-related injury, greater total ASI scores were significantly related to greater self-reported severity, but not frequency, of PTSD symptoms (Asmundson, Norton, Allderdings, Norton, & Larsen, 1998). Moreover, ASI total scores were significantly associated with each of the PTSD symptom clusters (i.e., reexperiencing, arousal, avoidance). Lang and colleagues (2002) also evaluated the relation between ASI factor scores (after controlling for depressive symptoms) and self-reported PTSD severity among women who had or had not experienced IPV. Here, only the Mental Incapacitation Concerns factor was significantly related (Lang et al., 2002). More recently, Keogh, Ayers, and Francis (2002) evaluated prospective associations between AS and PTSD symptoms among 40 women who underwent childbirth. Only the social and physical concerns factors were entered into a regression analysis (that controlled for general levels of emotional distress) predicting posttraumatic symptomatology, because the Mental Incapacitation Concerns factor was not correlated with PTSD symptoms. The social, but not physical, concerns factor emerged as a significant predictor, with higher levels predicting greater symptom development, and this pattern of results did not change when panic attack history was covaried.

Together, there is theoretical and empirical evidence that both the AS global construct and its factors are related to posttraumatic symptomatology. However, this literature is limited in that each study has relied on examination of the main effect of AS. That is, investigations have not addressed how AS may interact with other theoretically relevant factors in predicting posttraumatic stress symptomatology. Research suggests greater frequency of trauma exposure is associated with greater likelihood of developing PTSD (Messman & Long, 1996). Thus, examination of the interaction between AS and frequency of trauma exposure is a fruitful next step in improving the predictive ability of models aiming to predict PTSD symptomatology related to a traumatic event. With this background, the current study evaluated if AS moderates the relation between frequency of trauma exposure and posttraumatic stress responding among a sample of young adults. Conceptually, a moderator "affects the relationship between two variables, so that the nature of the impact of the predictor on the criterion variable varies according to the level of the moderator" (Holmbeck, 1997, p. 599). Statistically, after accounting for variance associated with the main effects of each predictor, the product term of these variables interacts to significantly predict the criterion variable. In the present case, then, the association between trauma exposure and posttraumatic stress responding would be expected to vary as a function of AS. Specifically, it was expected that greater trauma exposure frequency would be associated with higher levels of PTSD symptoms among individuals high in AS, whereas exposure frequency would have little effect on individuals low in AS. This prediction was based upon evidence suggesting traumatic life events may be particularly anxiety-provoking for individuals high in AS, as these persons may perceive such symptoms as relatively more aversive than their low AS counterparts.

Method

Participants

The sample consisted of 61 individuals (38 female; M_{age} = 21.15 years, SD = 5.16) screened from a larger sample of participants (n = 110) from an Appalachian university. Examining

risk-related processes relevant to PTSD within a rural sample is particularly important from a public health perspective in light of the significantly higher rates of anxiety and depression within this population as compared to urban populations (Beck, Jijon, & Edwards, 1996; Muntaner & Barnett, 2000). The inclusionary criterion for participation was having experienced at least one traumatic event, as indexed by the Posttraumatic Diagnostic Scale (PDS; Foa, 1995). The traumatic events most frequently reported by participants included serious accident, fire, or explosion (n = 26, 42%), natural disaster (n = 29, 47%), nonsexual assault by a family member or someone known (n = 14, 23%), nonsexual assault by a stranger (n = 12, 19%), sexual assault by a family member or someone known (n = 5, 8%), sexual assault by a stranger (n = 8, 13%), military combat (n = 3, 4%), sexual contact by someone more than 5 years older when the participant was younger than 18 years old (n = 15, 24%), imprisonment (n = 8, 13%), torture (n = 1, 1%), life-threatening illness (n = 17, 27%), and other (n = 17, 27%). Approximately 6% (n = 4) met full criteria for PTSD as indexed by the PDS (Foa, 1995). In terms of ethnicity, 88% were White, 5% African American, 3% Asian American, 1% other, and the remainder did not specify their ethnicity.

Measures

Anxiety Sensitivity Index (ASI). The ASI (Reiss et al., 1986) is a 16-item measure on which respondents indicate on a 5-point Likert-type scale (0 = "very little" to 4 = "very much") the degree to which they are concerned about possible negative consequences of anxiety-related sensations. Summing all responses derives the total ASI score; total scores range from 0 to 64. The ASI has high levels of internal consistency and good test-retest reliability (Peterson & Reiss, 1992). Research suggests that the ASI has three lower-order factors that all load on a single higher-order factor (see Zinbarg, Mohlman, & Hong, 1999, for a review). The lower-order factors represent physical, mental incapacitation, and social concerns, and the higher-order factor represents the global AS construct (Stewart, Taylor, & Baker, 1997). The ASI is unique from, and demonstrates incremental validity compared to, measures of negative affect, including trait anxiety (McNally, 1994).

Posttraumatic Diagnostic Scale (PDS). The PDS (Foa, 1995) is a 49-item self-report instrument designed to assess the presence of posttraumatic stress symptomatology, based on criteria outlined in the *Diagnostic and Statistical Manual of Mental Disorders–Fourth Edition* (DSM-IV; APA, 2000). The PDS is a well-established measure of trauma-related responding and has excellent psychometric properties (Foa, Cashman, Jaycox, & Perry, 1997). Respondents report if they have experienced any of 12 traumatic events including an "other" category (which are totaled to derive a total traumatic event exposure index) and then indicate which event disturbed them the most. Respondents then rate the frequency (0 = "not at all or only one time" to 3 = "five or more times a week/almost always") of 17 PTSD symptoms in relation to the most-disturbing event endorsed (total score range of 0 to 51). Individual items load on several subscales: (a) exposure to traumatic event and peritraumatic responding, (b) reexperiencing the event, (c) avoidance of cues associated with the event, and (d) arousal. The PDS can be utilized as a dichotomous (i.e., PTSD-positive vs. PTSD-negative) or continuous measure of symptomatology. In the current investigation, a sum of the number of traumatic events endorsed was utilized to index total trauma exposure frequency. The total symptom severity score (sum of frequency ratings of each symptom) was utilized as a continuous index of posttraumatic stress symptomatology. Higher scores on the PDS indicate more severe symptomatology.

Alcohol Use Disorders Identification Test (AUDIT). The AUDIT (Babor, de la Fuente, Saunders, & Grant, 1992) was constructed by World Health Organization investigators and is a widely used measure for the assessment of alcohol use and associated problems. There is a large body of evidence supporting the validity and utility of the AUDIT (e.g., Saunders, Aasland, Babor,

de la Fuente, & Grant, 1993). In the current investigation, items indexing total number of drinks consumed on a typical drinking occasion [rated on a scale from 0 ("less than 3") to 4 ("10 or more")] and frequency of drinking [rated on a 0 ("never") to 4 ("4 or more times a week") scale] were summed to create an index of total alcohol use.

Positive Affect Negative Affect Scale (PANAS). The PANAS is a mood measure commonly used in psychopathology research (Watson, Clark, & Tellegen, 1988). It assesses two global dimensions of affect: negative and positive. Only the negative affect scale (PANAS-NA) was used in this study. A large body of literature supports the reliability and validity of the PANAS (Watson, 2000).

Procedure

Participants were informed the purpose of the study was to learn how life events affect emotions. After providing written informed consent, participants completed the assessment instruments anonymously in an individual format during a single laboratory visit. The measures were randomly ordered so as to decrease the probability of response set biases. Participants were debriefed as to study objectives prior to their departure. All participants received course credit for their efforts.

Data Analysis

To address the hypotheses of the current investigation, hierarchical multiple regression analyses were performed to test the incremental (or relative) predictive validity (Haynes & Lench, 2003; Sechrest, 1963) of the AS (i.e., total and factor scores on the ASI) $\times$ total number of traumatic events interaction term. The criterion measure was total symptom severity scores on the PDS. Due to their theoretical significance and prior empirical demonstrations of significant associations with posttraumatic stress problems (see Brewin, Andrews, & Valentine, 2000; Kessler, Sonnega, Bromet, Hughes, & Nelson, 1995; Mineka, Watson, & Clark, 1998), the following covariates were employed to ensure any observed effects were not due to these factors: alcohol use, gender, family income, and negative affectivity. Predictor variables, specifically, were divided into three steps in the hierarchy: (a) covariates (i.e., alcohol use, gender, family income, and negative affectivity) were entered at step 1, (b) the main effects of AS and number of traumatic events were entered at step 2, and (c) the interaction term for AS and number of traumatic events interaction was entered at step 3. Main effect variables were mean-centered prior to computing the product term (Aiken & West, 1991). This model tests if AS moderates the relation between total number of traumatic events and posttraumatic stress symptomatology (Baron & Kenny, 1986), and ensures any observed effects for the interaction term are unique and cannot be attributed to shared variance with the other factors in steps 1 or 2 (Cohen & Cohen, 1983). Effect size was indexed by squared semipartial correlations (sr^2).

Results

Descriptive Data and Zero-Order Relations Among Theoretically Relevant Variables

See Table 1 for the means and standard deviations for, and correlations among, all relevant variables. Relations among predictor variables were examined first. Gender and family income were unrelated to predictor and criterion variables. Anxiety Sensitivity Index total, Mental Incapacitation Concerns, and Physical Concerns scores were significantly positively correlated with PANAS–Negative Affect scores, whereas ASI–Social Concerns scores were not significantly related. The ASI total and Physical Concerns scores were significantly positively associated with number of traumatic experiences, whereas ASI–Social Concerns scores were not significantly

TABLE 1. Descriptive Data and Zero-Order Relations Between Predictor and Criterion Variables

	n (%) or Mean (*SD*)	1	2	3	4	5	6	7	8	9	10
1. Volume of alcohol use	2.63 (2.21)	—	−.25	.12	−.00	.05	.02	−.00	.00	.00	−.00
2. Gender	38 (62%)	—	—	−.08	.21	.20	.17	−.17	.16	−.00	.10
3. Annual family income	2.33 (0.96)	—	—	—	−.08	−.06	−.21	.09	−.14	−.11	−.15
4. Negative affectivity	21.65 (7.02)	—	—	—	—	.44**	.43**	.21	.49**	.27*	.66**
5. AS—Mental incapacitation concerns	2.42 (3.34)	—	—	—	—	—	−.72**	.26*	.83**	.19	.56**
6. AS—Physical health concerns	8.73 (6.97)	—	—	—	—	—	—	.37**	.94**	.31*	.49**
7. AS—Social concerns	6.93 (2.19)	—	—	—	—	—	—	—	.53**	.12	.10
8. AS—Total score	20.15 (11.25)	—	—	—	—	—	—	—	—	.28*	.53**
9. Trauma exposure	2.55 (2.00)	—	—	—	—	—	—	—	—	—	.39**
10. PDS total symptom severity score	7.31 (11.44)	—	—	—	—	—	—	—	—	—	—

Note. $n = 61$; Volume of alcohol use: sum of the total number of drinks consumed on a typical drinking occasion and overall frequency of drinking; AS = Anxiety Sensitivity Index (Reiss et al., 1986); gender dummy coded as 1 = male, 2 = female; annual family income was dummy coded as 1 = less than $25,000, 2 = $25,000 to $50,000, 3 = $50,000 to $100,000, 4 = more than $100,000; *n* for subject gender reflects women; PDS = Posttraumatic Diagnostic Scale (Foa, 1995).

*$p < .05$. **$p < .01$. ***$p < .001$.

related. Mental Incapacitation Concerns scores were not significantly related to number of traumatic experiences.

Zero-order correlations also were computed to examine the pattern of relations among the predictor variables and posttraumatic stress symptomatology. In terms of covariates, only PANAS–Negative Affect scores significantly (positively) related to posttraumatic stress symptomatology. Consistent with hypotheses, ASI total, Mental Incapacitation Concerns, and Physical Concerns scores, as well as number of traumatic experiences, were significantly positively correlated with posttraumatic stress symptomatology. The Social Concerns factor was not significantly related to posttraumatic stress symptomatology.

Prediction of Posttraumatic Stress Symptomatology

See Table 2 for a summary of regression analyses. In the regression analysis that utilized ASI total scores, the predictor variables together explained 67% of the overall variance (adjusted $R^2 = .63$), $F(7, 52) = 15.37$, $p < .001$. At step 1 of the model, PANAS–Negative Affect scores were a significant predictor, such that higher levels of negative affectivity were associated with greater posttraumatic stress symptomatology. After controlling for alcohol use, gender, family income, and negative affectivity at step 1, number of traumatic events explained a unique 2% of variance, and ASI–total scores uniquely accounted for an additional 3%, with the entire model at step 2 accounting for 54% of total variance (adjusted $R^2 = .49$). At step 3, the number of traumatic events × AS total score interaction term significantly accounted for an additional 12% of unique variance.

In terms of analyses utilizing ASI factors, the first step of all models was comparable to that described for the regression utilizing the ASI total score (i.e., 46% of the variance accounted for by PANAS–Negative Affect scores, the only significant predictor). For the ASI–Mental Incapacitation Concerns factor, the total set of predictors significantly accounted for 68% of the variance (adjusted $R^2 = .64$), $F(7, 52) = 16.17$, $p < .001$. Number of traumatic experiences and Mental Incapacitation Concerns scores explained an additional 3% and 6% of variance, respectively, at step 2 of the model, increasing the overall variance accounted for to 58% (adjusted $R^2 = .53$). The interaction term (using ASI–Mental Incapacitation Concerns) at step 3 significantly explained an additional 10% of the variance in posttraumatic symptomatology. When ASI–Social Concerns scores were utilized, the entire model significantly accounted for 58% of the variance (adjusted $R^2 = .53$), $F(7, 52) = 10.56$, $p < .001$. At step 2 of the model, number of traumatic experiences significantly accounted for 4% of unique variance, increasing the model's explanatory ability to 51% (adjusted $R^2 = .46$) of the variance, whereas AS–Social Concerns was not a significant predictor. The interaction term (using AS–Social Concerns) significantly contributed 7% of unique explained variance to the model at step 3. When ASI–Physical Concerns scores were utilized, the entire model significantly explained a total of 67% of the variance (adjusted $R^2 = .62$), $F(7, 52) = 15.05$, $p < .001$, in posttraumatic symptomatology. Number of traumatic experiences and Physical Concerns scores each explained a unique 2% of the variance, increasing the model's explanatory ability at step 2 to 53% of the variance (adjusted $R^2 = .48$). The interaction at step 3, using ASI–Physical Concerns scores, significantly explained an additional 12% of unique variance.

The forms of the number of traumatic events × AS interactions predicting posttraumatic stress symptomatology were examined based on recommendations of Cohen and Cohen (1983, pp. 323, 419). Specific values for each predictor variable (one half of a standard deviation above and below the mean for number of traumas experienced and ASI scores) were inserted into the regression equation. The forms of the interactions supported hypotheses (see Figure 1). Specifically, among individuals high in AS, greater trauma exposure frequency was associated with higher levels of PTSD symptoms, whereas exposure frequency had little or no effect on individuals low in AS.

TABLE 2. INDIVIDUAL VARIABLE CONTRIBUTIONS PREDICTING POSTTRAUMATIC STRESS SYMPTOMATOLOGY

	ASI Total Score			Mental Incapacitation Concerns			Social Concerns			Physical Concerns		
	ΔR^2	β	sr^2	ΔR^2	β	sr^2	ΔR^2	β	sr^2	ΔR^2	β	sr^2
Step 1	.46***			.46***			.46***			.46***		
Alcohol use		−.02	.00		−.02	.00		−.02	.00		−.02	.00
Gender		−.05	.00		−.05	.00		−.05	.00		−.05	.00
Family income		−.10	.01		−.10	.01		−.10	.01		−.10	.01
Negative affectivity		.67***	.42		.67***	.42		.67***	.42		.67***	.42
Step 2	.07*			.11**			.04			.06*		
Alcohol use		−.02	.00		−.04	.00		−.03	.00		−.03	.00
Gender		−.05	.00		−.07	.00		−.05	.00		−.05	.00
Family income		−.06	.00		−.07	.00		−.07	.00		−.05	.00
Negative affectivity		.51***	.18		.48***	.16		.63***	.32		.54***	.22
Trauma exposure		.18	.02		.19*	.03		.22*	.04		.18	.02
AS		.21	.03		.30**	.06		−.06	.00		.18	.02
Step 3	.13***			.10***			.07**			.13***		
Alcohol use		.01	.00		.01	.00		−.02	.00		.02	.00
Gender		−.12	.01		−.12	.01		−.09	.00		−.11	.01
Family income		−.09	.00		−.12	.01		−.08	.00		−.09	.00
Negative affectivity		.54***	.20		.53***	.20		.58***	.27		.56***	.23
Trauma exposure		−.08	.00		−.04	.00		.08	.00		−.09	.00
AS		.13	.01		.26**	.04		−.02	.00		.11	.00
AS × traumas[a]		.47***	.12		.40***	.10		.31**	.07		.48***	.12

Note. $n = 61$; β = standardized beta weight; gender dummy coded as 1 = male, 2 = female; annual family income was dummy coded as 1 = less than $25,000, 2 = $25,000 to $50,000, 3 = $50,000 to $100,000, 4 = more than $100,000; AS = anxiety sensitivity.

[a]Interaction term for Anxiety Sensitivity Index (Reiss et al., 1986) scores and total number of self-reported traumatic experiences.

Discussion

There is growing recognition that AS is relevant to vulnerability processes for anxiety and its disorders. Although previous work has indicated AS is related to PTSD symptomatology, no work has addressed whether this cognitive risk factor moderates the effect of other established PTSD risk factors. To begin to fill this gap in the existing literature, the purpose of the present study was to test the hypothesis that AS moderates the effect of frequency of traumatic event exposure on PTSD symptoms.

As predicted, among individuals high in AS, greater trauma exposure frequency was associated with higher levels of PTSD symptoms, whereas exposure frequency had little or no effect on individuals low in AS. Importantly, these significant effects (ranging in effect size from 7% to 12% of unique variance) were above the variance accounted for by theoretically relevant covariates (i.e., negative affectivity, alcohol use, family income, and gender) as well as the respective main effects. Thus, there is broad-based consistency in this cross-sectional study that AS global and lower-order facets are important cognitive factors in terms of better understanding the relation between frequency of traumatic event exposure and PTSD symptoms among young adults. The fact that the AS $\times$ frequency of trauma interaction effects were over and above negative affectivity, in particular, suggests that the present relations cannot be attributed to simply a generalized tendency to experience negative emotional symptoms. Future scientific attention should be devoted to clarifying the mechanisms underlying the observed association. For instance, a potential mechanism of action underlying the observed pattern of findings is that high levels of AS and prior trauma exposure may function to increase affective sensitivity to a traumatic event, thereby increasing symptomatic responding via increasing the severity of a traumatic experience. That is, a person high in AS who has frequently experienced trauma, relative to a person low in either of these variables, may experience greater fear-related reactivity during a traumatic event. This could result in greater conditioning of trauma-related cues and thus avoidance of, and hyperarousal in response to, such cues. Clearly, this theory-driven hypothesis requires future empirical examination.

A significant main effect of number of traumatic events, beyond variance accounted for by the covariates, also was observed. This extends previous demonstrations suggesting that increased trauma exposure increases the likelihood of developing PTSD (Messman & Long, 1996). In regard to AS, only the Mental Incapacitation Concerns lower-order facet emerged as a significant predictor. These data are consistent with findings from research with women survivors of IPV (Lang et al., 2002), yet inconsistent with research on women who underwent childbirth (Keogh et al., 2002). One possible explanation for this pattern is that fears of mental incapacitation are more directly related to IPV than childbirth. Given that participants in the present investigation were not selected on the basis of a particular type of trauma, future work may seek to identify a population that has been exposed to only one type of theoretically relevant traumatic life event and evaluate the extent of AS specificity in that context (e.g., burn victims and AS Physical Concerns). Second, it is noteworthy that AS shared a range of 4% to 24% of variance with negative affectivity (see Table 1). These data empirically demonstrate that AS is not equivalent to a generalized tendency to experience negative affect, and moreover, accounts for unique variance above such a disposition. Given that previous work has found AS is malleable (Telch et al., 1993), it may represent an ideal target for future PTSD prevention and intervention work.

There are a number of interpretive caveats and directions for future study that warrant comment. First, the present study employed a cross-sectional design. Although such an approach is a useful and efficient starting point for evaluating the potential value of AS in relation to PTSD, it is limited. Specifically, it provides a "snapshot" of associations rather than a window into their nature across time. Future work would be well-served to replicate and extend the present findings using prospective methodologies that can elucidate the extent to which the

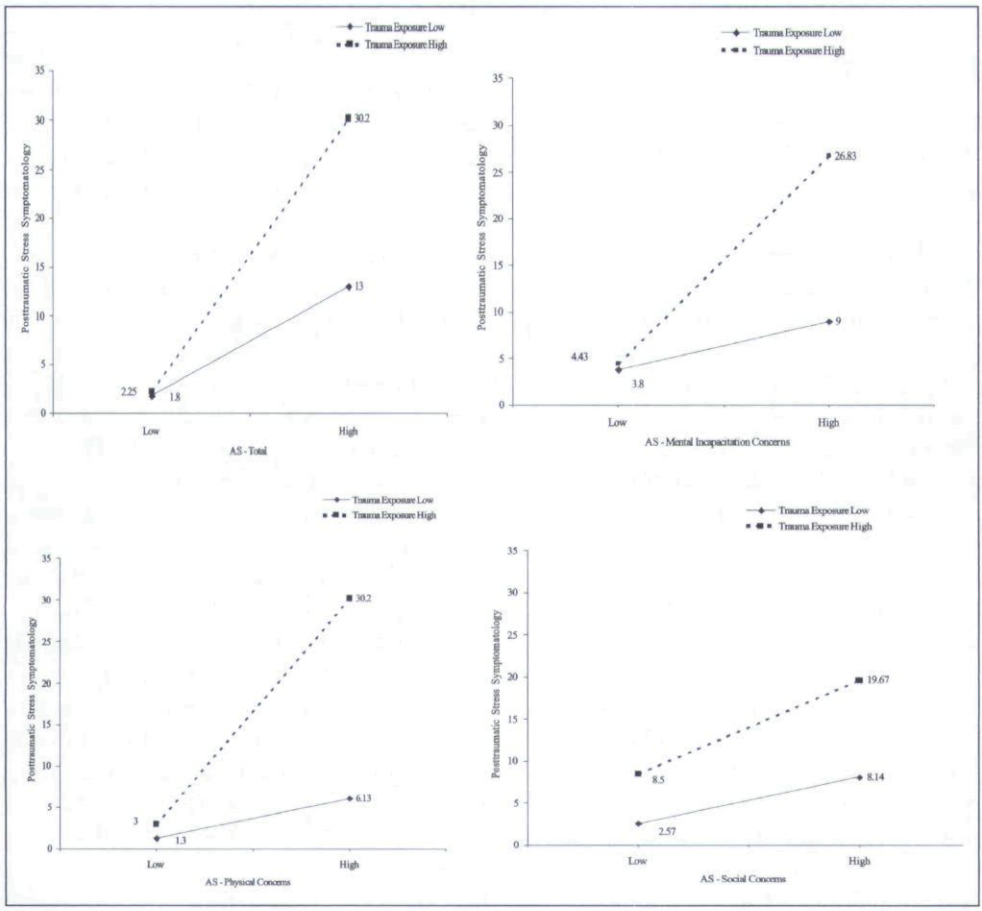

FIGURE 1. Posttraumatic stress symptomatology, as indexed by symptom severity scores on the PDS (Foa, 1995), as a function of Anxiety Sensitivity Index (Reiss et al., 1986) total and factor scores and total number of self-reported exposures to traumatic events (Foa, 1995) among participants one-half of a standard deviation above and/or below the mean for each predictor.

observed relations are apparent over time. Second, the present data, similar to all of the previous AS-trauma research, are correlational by virtue of the methodological design employed. Thus, the directional nature of the observed effects cannot be explicated. Evidence suggests AS may exacerbate PTSD symptoms (Keogh et al., 2002), but it also is possible traumatic life events can increase AS. That is, there are likely bidirectional influences between affect-relevant individual difference factors like AS and trauma. If AS effects are further delineated in prospective work, it may be useful to experimentally target this factor in prevention work using psychosocial treatment strategies (see Feldner, Zvolensky, & Schmidt, in press, for a discussion). Such work would be useful in developing theoretically driven preventive interventions for PTSD in general, but would simultaneously more firmly establish the role of AS in the nature of this condition. Third, the present study did not rule out preexisting psychopathology. Although this issue is not unique to extant AS-PTSD work, it is nonetheless a potential caveat as variability in AS likely is associated with various anxiety phenotypes (i.e., AS would be correlated with anxiety disorders). Thus, it would be important to document AS effects on PTSD symptoms among

persons without a history of other preexisting psychopathology, including nonclinical panic attacks, as indexed by psychiatric screenings.

Fourth, future research would enhance confidence in the current conclusions by incorporating behavioral tests of PTSD symptomatology (e.g., behavioral avoidance tests) and constructs related to AS (e.g., reactivity to biological challenges). Finally, the decision to utilize a wide array of traumatic events may have obscured differential relations between AS and specific types of trauma. For instance, AS may play a more direct role in responsivity to a traumatic event that is longer in duration, and therefore characterized by greater periods of anxiety-related sensations (e.g., rape), than a brief trauma characterized by an abrupt fear response with relatively less opportunity for extended anxiety (e.g., tornado). To test if AS differentially relates to posttraumatic symptomatology as a function of trauma type, future research could more specifically select traumas (e.g., only sexual assault) and examine the moderating role of AS in the specific trauma exposure–posttraumatic symptom association.

In summary, the current investigation adds to a growing literature that suggests AS is related to PTSD symptoms. Further investigation of the likely complex interplay among individual difference factors and aspects of traumatic experiences will aid in the continued refinement of etiological theories of PTSD as well as preventive efforts based on such theories.

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Offprints. *Requests for offprints should be directed to Matthew T. Feldner, Assistant Professor, Department of Psychology, University of Arkansas, Fayetteville, AR 72701. E-mail: mfeldne@uark.edu*

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